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Natriuretic peptides increase a K⁺ conductance in rat mesangial cells.

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Mesangial cells (MC) are a main target of natriuretic peptides in the kidney and are thought to play a role in regulating glomerular filtration rate. We examined the influence of cGMP-generating (i.e. guanosine 3',5'-cyclic monophosphate) peptides on membrane voltages (V_m) of rat MC by using the fast whole-cell patch-clamp technique. The cGMP-generating peptides were tested at maximal concentrations ranging from 140 to 300 nmol/l. Whereas human CNP (C natriuretic peptide), rat guanylin and human uroguanylin had no significant effect on V_m these cells, human BNP (brain natriuretic peptide), rat CDD/ANP-99-126 (cardiodilatin/atrial natriuretic peptide) and rat CDD/ANP-95-126 (urodalin) hyperpolarized V_m significantly by 1.6 ± 0.4 mV (BNP, $n=8$), 3.7 ± 0.3 mV (CDD/ANP-99-126, $n=25$) and 2.8 ± 0.4 mV (urodalin, $n=9$), respectively. The half-maximally effective concentration (EC_{50}) for the latter two was around 400 pmol/l each. This hyperpolarization could be mimicked with 0.5 mmol/l 8-bromo-guanosine 3',5'-cyclic monophosphate (8-Br-cGMP) and was blocked by 5 mmol/l Ba^{2+} . The K⁺ channel blocker 293 B (100 micromol/l) depolarized basal V_m by 4.3 ± 0.4 mV ($n=8$), but failed to inhibit the hyperpolarization induced by CDD/ANP-99-126 (160 nmol/l) ($n=8$). The K⁺ channel opener cromakalim (10 micromol/l) neither influenced basal V_m nor altered the hyperpolarization induced by 160 nmol/l CDD/ANP-99-126 ($n=8$). Adenosine (100 micromol/l) hyperpolarized V_m by 13.4 ± 1.3 mV ($n=16$). At 100 micromol/l, 293 B did not inhibit the adenosine-induced hyperpolarization ($n=6$). At 160 nmol/l, CDD/ANP-99-126 enhanced the adenosine-induced hyperpolarization significantly by 1.5 ± 0.6 mV ($n=10$). CDD/ANP-99-126 (160 nmol/l) failed to modulate the value to which V_m depolarized in the presence of 1 nmol/l angiotensin II ($n=10$), but accelerated the repolarization to basal V_m by $49 \pm 20\%$ ($n=8$). These results indicate that the natriuretic peptides CDD/ANP-99-126, CDD/ANP-95-126 and BNP

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hyperpolarize rat MC probably due to an increase of a K⁺ conductance. This effect modulates the voltage response induced by angiotensin II. The natriuretic-peptide-activated conductance can be blocked by Ba²⁺, but not by 293 B and cannot be activated by cromakalim. This increase in the K⁺ conductance seems to be additive to that inducible by adenosine, indicating that different K⁺ channels are activated by these hormones.

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